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A Comparative Study of Fractional CO_2 Laser Mediated Topical Triamcinolone Versus Intralesional Acetonide in the Treatment of Keloid Scars

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Abstract

Objective: Pathological scars, particularly keloids, cause significant physical and emotional distress. This study compared the effectiveness, safety, and patient satisfaction of fractional CO_2 laser with topical triamcinolone versus intralesional triamcinolone acetonide (ILTA).

Methods: This prospective interventional study included 24 patients with keloids. Group A received fractional CO_2 laser followed by topical triamcinolone, while Group B received intralesional triamcinolone acetonide (ILTA, 10–40 mg/mL). Both groups underwent two treatment sessions, with scars assessed using the Vancouver Scar Scale (VSS) and Patient and Observer Scar Assessment Scale (POSAS) at baseline, during treatment, and 3 months post-treatment.

Results: Both treatments produced significant clinical improvement (VSS, $P < 0.001$), with no significant difference between groups ($P > 0.05$). However, POSAS scores significantly favored fractional CO_2 laser with topical triamcinolone ($P < 0.05$), indicating better scar pliability, symptom relief, and patient satisfaction.

Conclusion: Ablative Fractional CO2 laser with topical triamcinolone demonstrates comparable clinical efficacy to conventional intralesional steroid therapy in keloid management. Notably, the laser-assisted approach offers superior patient tolerability and satisfaction, making it a valuable, minimally invasive alternative.

Keyword: Keloid; Fractional CO2 laser; Triamcinolone acetonide; Laser-assisted drug delivery; Intralesional injection.

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Introduction

Keloids are a fibro proliferative condition in the dermis lacking malignant potential that usually arises after minor skin injuries or slight trauma in those with a genetic predisposition. Keloids show a distinct tendency to occur in particular anatomical sites, such as the parasternal region, earlobes, deltoid areas, and the area behind the ears. They are more prevalent in

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Asians and blacks, with incidence ratios of 5:1 and 15:1, respectively, compared to Caucasians. People with HLA B14, BW16, DR5, and blood type A have a higher likelihood of developing keloids.[1] The distribution of keloids is equal in both sexes, and the highest incidence of the scar occurs in the second and third decades of life. It appears as fixed, irregular, mildly tender, and pink to purple in color with well-circumscribed margins and a shiny surface with occasional telangiectasia.[2] Pruritic keloids are distinguished histologically by a significantly higher concentration and density of dermal mast cells containing dense secretory granules compared to non-itchy lesions.[3]

The abnormal wound healing in keloids is marked by prolonged inflammation, active fibroblasts, and a disruption in the balance of collagen production and breakdown. The expression levels of matrix metalloproteinases (MMPs) and their inhibitors, referred to as tissue inhibitors of metalloproteinases (TIMPs), are modified. In keloids, MMP-1, MMP-2, MMP-13, TIMP-1, and TIMP-2 levels are elevated, whereas MMP-3 levels are lower in comparison to normal scars. These alterations lead to the abnormal collagen accumulation and heightened type I/III collagen ratio seen in keloid tissue.[4] TGF-β and histamine encourage dermal fibroblasts to generate periostin, a matricellular protein that plays a role in tissue remodelling and is elevated in diseased scar tissue relative to normal skin. Periostin is recognized as a pruritogen that directly stimulates itch through integrin receptors in nerve fibers and triggers itch through the Th2 cytokine pathway.[3] Keloid scars demonstrated normal thickness of the epidermis. The papillary dermis shows scarring with the presence of large, broad, glassy, eosinophilic, focally fragmented, and haphazardly arranged collagen complexes referred to as "keloid collagen," which occupied the full thickness of the reticular dermis and showed positivity for α-SMA expressing myofibroblasts in one third of keloid scars. The presence of horizontal fibrous bands with an advancing edge underneath the epidermis, described as "pseudopodia-like extensions". Prominent telangiectasia in the papillary dermis and vertically oriented blood vessels have been reported.[5]

Prevention includes

A- Tension-Free Primary Closure Rapid primary closure using tension-free techniques is very important for minimizing the effects of bad scars.[6]

B- Passive Mechanical Stabilization It has been applied using silicone sheets providing occlusion and hydration of the scar surface. Silicone sheeting is recommended for use from two weeks after primary wound treatment for more than 12 hours a day for at least two months.[6]

C- Flavonoids or bioflavonoids Quercetin exerts antifibrotic actions that may be mediated through the induction of MMP-1 or the inhibition of SMAD2, SMAD3, or SMAD4 expression. The instructions as follows: start two weeks after primary wound treatment, and twice daily for four to six months.[6]

Treatment

Corticosteroids Corticosteroids have been shown to make keloids smaller by working in ways inside the body. At first they lower swelling by stopping blood cells from becoming active and by getting in the way of how monocytes move and eat things. Next they are very good at narrowing blood vessels, which means less oxygen and food reach the wound. Then they stop cells from dividing, which stops skin cells and connective tissue cells from growing. This makes it harder for the skin to heal and for new collagen to form. Also steroids seem to lower the amount of proteins that stop enzymes from breaking down collagen. This means the body's own collagen-dissolving enzymes can work better. TAC also lowers the levels of alpha-1-antitrypsin and alpha-2-macroglobulin. These are usually higher in tissue and help stop collagen from breaking down. Corticosteroids change how fibroblasts grow and make things. This leads to their breakdown. Also less TGF-β,

IGF-1 and hydroxyproline were seen in scars that were treated with methylprednisolone. Studies found that dexamethasone made keloids get smaller by linking with receptors in the body. This stopped the body from making VEGF. Stopped fibroblasts from growing. The use of TAC (Kenalog) at concentrations from 10 to 40 mg/mL was based on how big and where the keloid was. For keloids, on the chest or arms the treatment usually started at 40 mg/mL. and changed accordingly at subsequent visitsside effects, including local issues like telangiectasias, atrophy of skin and subcutaneous fat, pigmentary alterations (hypopigmentation and hyperpigmentation), skin necrosis, and ulcerations, as well as systemic effects, such as Cushing's syndrome.[7]

Topical imiquimod Imiquimod 5% cream, a topical immune-response modifier, Post-surgical use stimulates interferon, a proinflammatory cytokine, which increases collagen breakdown.[8]

A- Topical mitomycin C It has been shown to inhibit fibroblast proliferation and decrease scar formation both in vivo and in vitro.[9]

B- Bleomycin It causes a reduction in lysyl-oxidase levels, which is a cross-linking enzyme involved in collagen maturation. The most commonly encountered side effects include pain, superficial ulceration at the sites of injection, transient hyperpigmentation, and dermal atrophy.[9]

C- Intralesional 5-fluorouracil (5-FU) It inhibits fibroblast proliferation. Also has an inhibitory effect on TGF-β. It is effective as intralesional, given either once weekly or once every 2 weeks. Common adverse effects include: pain at the injection site, ulceration, burning, and hyperpigmentation.[9]

Interferons INF-α2b and INF-γ have been demonstrated to reduce collagen production and induce TGF-β1 down-regulation.[10]

Verapamil Verapamil stimulates the production of procollagenase in keloids and regular human cultured fibroblasts, leading to decreased fibrous tissue formation.[10]

Intralesional Methotrexate (IL-MTX) IL-MTX and triamcinolone are an effective mode of therapy for keloids and were much better than intralesional injection of MTX or TAC alone.[11]

Botulinum Toxin Its capacity to reduce muscle tension, stop fibroblast cell cycles in the non-proliferative phase, and adjust TGF-β1 expression.[4]

Cryosurgery It causes destruction of keloid scars through direct cell anoxia and vascular injury, and ultimately tissue necrosis and sloughing. Side effects include infection, sloughing, transient hypo/depigmentation, transient hyperpigmentation, atrophy, and scarring.[12]

Surgical therapy Keloid excision may trigger collagen production, heightening the chances of recurrence and possibly resulting in a keloid larger than the initial one; therefore, adjunct treatments such as radiation therapy should consistently accompany surgery to reduce recurrence rates.[4]

Radiation Surgical excision followed by radiation has become a mainstay of keloid treatment in an effort to prevent postoperative recurrence. The reduction of fibroblast proliferation and inhibition of collagen synthesis through downregulation of TGF-beta and mast cell histamine release is considered the fundamental mechanism of action.[13]

Lasers used in the treatment of keloids The mechanism of lasers is based on the concept of "selective photothermolysis. Ablative laser devices consist of fractional CO2 and 2940 nm Er-YAG. They lead to scar reduction by targeting the water in these tissues, reducing the size of the lesion. Nonablative lasers (such as 585 nm pulsed dye laser, 1064 nm Q-switched Nd-YAG laser, and 1064 nm long-pulsed Nd-YAG) can destroy the capillaries through the absorption of laser light energy by blood inside the

veins. The destruction of blood vessels causes hypoperfusion with a consequent reduction in oxygenation. Changes occur in the tissues as a result of the hypoxia, with the production of new collagen.[14]

Fractional ablative lasers Fractional ablative lasers, including both CO2 and Er: YAG, have combined the impressive results of ablative lasers with the lesser risk profile of nonablative lasers.[15]

Laser-assisted topical drug delivery Fractional ablative lasers create vertical microchannels introduced into the skin to facilitate topical drug delivery.[16] The notable impact of LADD is evident with hydrophilic substances (liquid and gel formulations), and applying topically within 6 hours after laser use enhances absorption, proving most effective in the first 30 minutes. Nonetheless, there is no rise in absorption noted when the product is used after 24 hours.[17]

Once the laser beam breaks through the epidermis and forms channels, drugs may disperse within the dermis, and inappropriately high energy may stimulate the proliferation of keloids, while relatively conservative low energy is more suitable for keloid management.[18]

Patient and Methods

This prospective, interventional, open therapeutic study was conducted at the Center of Dermatology and Venereology, Medical City, Baghdad, Iraq, from August 2023 to September 2025. The Institutional Review Board approved the protocol, and written informed consent was gathered from all patients or their parents if they were under 18 years

The study enrolled 24 patients presenting with clinical features of keloid scars of any size and duration. Eligible participants were males or females aged older than 14 years with any Fitzpatrick skin type. Exclusion criteria comprised receiving any keloid treatment within the preceding six months, pregnancy or lactation, psychologically unstable status, or active dermatological diseases such as psoriasis or vitiligo

Treatment Protocol

A within-patient split-lesion design was utilized. In 14 patients presenting with a single keloid, the lesion was divided into two equal halves using a surgical skin marker and ruler. In the remaining 10 patients with multiple keloids, separate individual scars were allocated to different treatment regimens

Group A (Laser-Assisted Drug Delivery): The designated site received fractional carbon dioxide laser therapy followed immediately by the topical application of triamcinolone acetonide suspension

Group B (Intralesional Injection): treated with standard intralesional triamcinolone acetonide injection alone

Before each session, topical anesthesia was achieved using EMLA cream applied under occlusion for one hour. Laser parameters were standardized for all patients: energy level of 12 mJ, 5 points/shot, pulse width of 3 ms, pitch distance of 0.7 mm, delivered via a single pass in a random scanning mode. For Group A, topical triamcinolone acetonide (40 mg/mL) was applied to the laser-treated area within 30 minutes to ensure maximum channel patency, followed by an occlusive film dressing for 6 hours. For Group B, intralesional triamcinolone acetonide was injected directly into the core of the lesion at a concentration of 10–40 mg/mL, titrated according to lesion size and anatomical location. Both groups received a total of two sessions at a 1-month interval.

Clinical Evaluation

Clinical assessments were performed at baseline, monthly before each session, and at a final follow-up visit three months after the last treatment. Lesion progress was quantitatively documented using standardized photography under identical lighting and distance. Objective scar severity was scored by Vancouver Scar Scale (VSS), which assesses four unique parameters: vascularity (0-3), pigmentation

(0-2), pliability (0-5), and height (0-3), resulting in a total score spectrum of 0 to 13.

The POSAS consists of two independent components: the Patient Scale and the Observer Scale. The Patient Scale captures six items completed directly by the participant: pain, itching, color, stiffness, thickness, and irregularity. The Observer Scale evaluates six analogous physical parameters assessed by a blinded dermatologist: vascularity, pigmentation, pliability, thickness, relief, surface, and overall opinion. Each item is scored from 1 (normal skin) to 10 (most severe scar possible), providing a cumulative score range of 7 to 70 for each scale. Safety was monitored by documenting all localized or systemic adverse effects at every follow-up visit.

Statistical analysis was performed with IBM SPSS Statistics software. Quantitative variables were represented as mean standard deviation (SD) and range, while categorical variables were shown as frequencies and percentages. Comparisons within the group tracking changes from baseline over time were analyzed using the paired t-test. Intergroup differences between the two therapeutic modalities at specific time points were evaluated using the independent Student's t-test or paired t-test where appropriate. Associations involving multi-categorical clinical parameters were analyzed using Variance Analysis (ANOVA). Statistical significance was determined as a p-value less than 0.05.

Results

A total of 24 patients with keloids were included in the study. There were 12 males and 12 females. The mean age was 28.1±8.9 years, while the mean duration of the keloid lesions was 5.0±3.0 years. Most patients had Fitzpatrick skin types III or IV. A positive family history of keloids was present in 25% of the patients. Other baseline characteristics are shown in **Table 1**.

The baseline VSS scores were similar at the two treatment sites. A gradual reduction in VSS was observed with both treatment approaches during treatment and follow-up. After the first treatment session, the intralesional steroid site showed a greater reduction in VSS compared with the CO_2 laser-assisted topical steroid site, and this difference remained statistically significant after the second session and at the final follow-up. When the response was assessed as percentage improvement from baseline, the difference between the two treatment sites was significant after the first session. This difference was no longer statistically significant after the second session or at the final follow-up. The VSS findings and percentage changes from baseline are summarized in **Tables 2 & 3**.

Table 1 Characteristics of study sample.

Outcome	*No. (n)*	*%age*
Age (years)		
<20years	3	12.5
20-29	12	50.0
30-39	5	20.8
≥40years	4	16.7
Mean±SD (Range)	28.1±8.9	(15.0-49.0)
Sex		
Male	12	50.0
Female	12	50.0
Family history		
Yes	6	25.0
No	18	75.0
Duration (years)		
<5years	13	54.2
5-9	7	29.2
≥10years	4	16.7
Mean±SD (Range)	5.0±3.0	(1.0-10.0)
Skin type		
Type II	3	12.5
Type III	11	45.8
Type IV	10	41.7

POSAS scores decreased progressively at both treatment sites over the course of treatment and follow-up.

Patient scores did not differ significantly between the

Table 2 Comparison between study patients by total VSS

	VSS CO2 +steroid	*VSS steroid*	*P value*
Pre VSS	9.3±1.6 (5.0-11.0)	8.9±1.7 (5.0-12.0)	0.083
Post 1 VSS	7.8±1.2 (5.0-10.0)	7.0±1.5 (2.0-9.0)	0.001[#]
Post2VSS	6.2±1.3 (4.0-9.0)	5.7±1.4 (2.0-8.0)	0.047[#]
Follow up	5.5±1.5 (2.0-8.0)	5.0±1.4 (2.0-7.0)	0.015

[#]: statistically significant difference ($P < 0.05$).

Table 3 Comparison between study patients by total VSS.

	Percentage improvement ±SD From baseline		*P value*
Sessions	*CO2+ steroid*	*Steroid*	*P value*
After 1st session	16.1% ±9.5	22.1%±11.5	0.018#
After 2nd session	32.8% ±13.4	35.5%±12.9	0.322
Follow up	40.5% ±15.5	44.1%±12.8	0.097

#: statistically significant difference ($P < 0.05$).

Table 4 Comparison of POSAS over time between CO2 laser-assisted topical steroid site and intralesional steroid site

	POSAS		*P value*
	CO2+Steroid site	*Steroid site*	
Patient Pre	38.9±6.5 (28-50)	38.8±6.2 (25-52)	0.824
Post 1	30.5±6.2 (20-42)	30.3±5.9 (20-44)	0.627
Post 2	24.5±5.2 (15-36)	23.9±4.5 (15-30)	0.110
Final	20.6±3.7 (15-28)	23.1±3.8 (15-29)	0.0001#
Observer Pre	39.9±5.4 (34-52)	39.7±5.1 (33-50)	0.653
Post 1	33.9±6.1 (25-48)	33.3±5.9 (25-46)	0.179
Post 2	27.0±5.8 (18-38)	26.6±5.5 (18-39)	0.499
Final	24.5±5.6 (17-37)	26.3±5.5 (18-39)	0.002#

#: statistically significant difference (P < 0.05).

two treatment sites at the earlier assessments. At the final assessment, however, the patient-reported POSAS score was significantly lower at the CO_2 laser-assisted topical steroid site. A similar pattern was observed with the observer scores. No significant difference was found between the two treatment sites during the earlier assessments, whereas a significant difference was observed at the final assessment, with lower scores at the CO_2 laser-assisted topical steroid site. The POSAS results are presented in **Table 4**.

Both treatment modalities were generally well tolerated. Following CO_2 laser-assisted topical steroid treatment, transient erythema and burning were the most commonly reported adverse effects. One patient developed localized post-inflammatory hyperpigmentation, which resolved during follow-up. Among the sites treated with intralesional steroid, localized hypopigmentation, telangiectasia, and exacerbation of localized acne vulgaris were observed. No serious adverse events or systemic complications were reported during the study. Representative clinical photographs illustrating the clinical response of keloid scars to both treatment modalities at baseline, after the first and second treatment sessions, and at follow-up are shown in **Figures 1** and **2**.

Discussion

The effectiveness of topical steroid delivery aided by fractional CO2 laser may be explained by the creation of microscopic thermal zones that enhance transdermal drug penetration while simultaneously inducing dermal remodeling.[18]

In the present study, both treatment groups showed statistically significant improvement in keloid severity over time, with a reduction in VSS scores from baseline to follow-up in both groups. Similar finding reported by Tawaranurak *et al*. with no statistically significant difference observed between the groups at the 1-year treatment interval.[19] Comparable findings have also been reported in Abd El-Dayem *et al*. who observed significant improvement in VSS scores on both laser-assisted topical steroid and intralesional steroid sides, showing no statistically meaningful difference between the two, while patients favored the laser-assisted topical approach due to reduced pain.[20] Park *et al*. also reported similar VSS improvement with both modalities, but markedly lower pain scores and higher patient preference for topical laser-assisted steroid delivery.[21] Regarding the intralesional triamcinolone injection (group B), a higher percentage of improvement from baseline than in group A. However, the difference did not show a statistically significant difference from the other group (p=0.097). These findings suggest that intralesional steroid injection may achieve more objective scar regression, particularly in thickness and hypertrophy, due to higher localized drug concentration delivered directly into the fibrotic tissue. These results are consistent with those reported by Behrangi *et al*.[22] who found that hypertrophy improved more significantly with intralesional triamcinolone compared with ablative fractional CO2 laser–assisted topical steroid therapy despite superior improvement in dyschromia and texture in the combination group. Regarding the POSAS scale, the present study demonstrated more

favorable outcomes for fractional CO_2 laser–assisted topical triamcinolone acetonide at the final follow-up visit. The discrepancy between VSS outcomes and patient- or observer-based assessments reflects the multidimensional nature of keloid evaluation. While the VSS primarily assesses physical characteristics

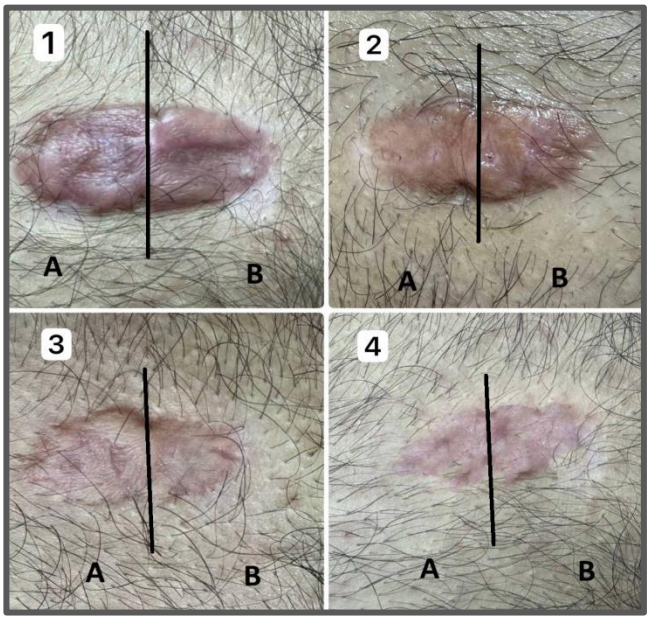

Figure 1 22-year-old male with a keloid on the chest for 5 years; 1: at baseline, 2: after 1st session, 3: after 2nd session, 4: follow-up (group A: CO2+ steroid, group B: intralesional steroid alone).

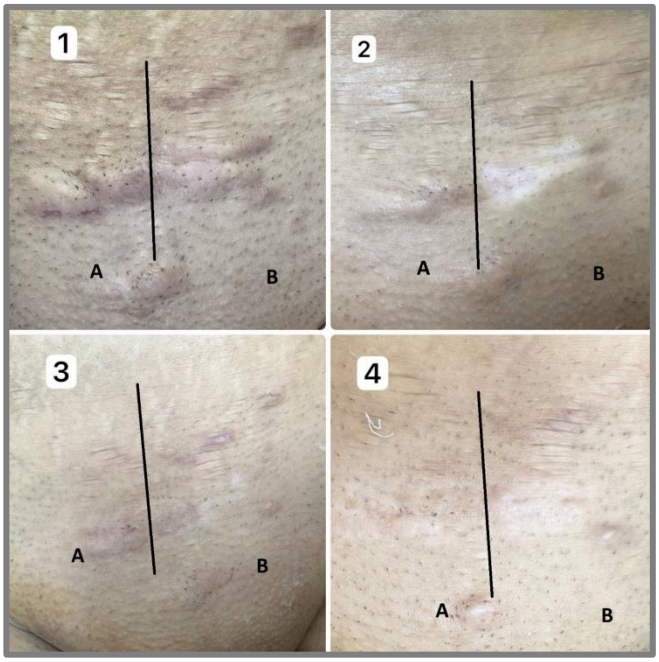

Figure 2 35 yrs old female with a keloid on the lower abdomin 2 years' duration; 1: at baseline, 2: after 1st session, 3: after 2nd session, 4: follow-up (group A: CO2+ steroid, group B: intralesional steroid alone).

such as scar height and pliability, POSAS incorporates symptoms, cosmetic appearance, and overall satisfaction, which may explain differences in perceived improvement. In the study by Wang *et al.*[18] significant improvement in POSAS scores was evident as early as one month following treatment, supporting the early clinical efficacy of fractional CO_2 laser–assisted topical triamcinolone. Moreover, the sustained reduction in both patient and observer scores at 24-month follow-up highlights the long-term durability of this therapeutic approach. Several studies have shown superior outcomes when the fractional CO2 laser is combined with intralesional steroid injection rather than topical application. Alexander *et al*. reported significantly greater improvement in keloids treated with fractional CO_2 laser plus intralesional TAC compared with intralesional TAC alone.[23] Likewise, Shafqat *et al*. demonstrated higher efficacy in patients receiving combined fractional CO2 laser and intralesional TAC compared with intralesional TAC monotherapy.[24] These findings suggest that the addition of laser therapy may potentiate the effects of intralesional steroid injection; however, they address a different therapeutic comparison than that evaluated in the present study.

Limitations of the Study

Sample Size Small cohort (n=24) from a single center, limiting broad generalizability.

Follow-up Relatively short post-treatment monitoring period to definitively track long-term recurrence rates.

Heterogeneity Inclusion of diverse anatomical sites and combination of both split-lesion designs and full-lesion treatments that could potentially impact the overall homogeneity of our statistical analysis.

Discussion

Both therapeutic protocols demonstrate significant clinical efficacy in managing keloid scars, achieving comparable objective regression by the conclusion of the treatment course.

Intralesional triamcinolone acetonide injections provide a more rapid initial reduction in scar height and hypertrophy, though this objective advantage narrows over subsequent sessions.

Fractional laser-assisted topical steroid application yields significantly higher patient satisfaction and superior POSAS scores due to reduced procedural pain and effective symptomatic relief.

The safety profile of laser-assisted drug delivery is superior to intralesional injection, causing fewer steroid-related complications such as tissue atrophy, persistent hypopigmentation, and telangiectasia.

Recommendations

Fractional laser-assisted topical steroid delivery should be prioritized for keloids located in cosmetically sensitive or highly visible anatomical regions to minimize the risks of persistent injection-induced atrophy or hypopigmentation.

Standard intralesional steroid injections should be reserved for thick, dense keloids located in hidden anatomical areas where immediate structural debulking is the primary clinical objective.

Clinical research should employ larger patient sample sizes and prolonged post-treatment follow-up durations to comprehensively monitor long term recurrence rates.

References

1. Nangole FW, Agak GW. Keloid pathophysiology: fibroblast or inflammatory disorders? JPRAS Open. 2019;22:44–54. doi: 10.1016/j.jpra.2019.09.004.
2. Mari W, Alsabri SG, Tabal N, Younes S, Sherif A, Simman R. Novel insights on understanding of keloid scar: article review. *J Am Coll Clin Wound Spec*. 2015;**7(1-3)**:1–7. doi: 10.1016/j.jccw.2016.10.001.
3. Hawash AA, Ingrasci G, Nouri K, Yosipovitch G. Pruritus in keloid scars: mechanisms and treatments. *Acta Derm Venereol*. 2021;**101(10)**:adv00578. doi: 10.2340/00015555-3943.
4. Kim HJ, Kim YH. Comprehensive insights into keloid pathogenesis and advanced therapeutic strategies. *Int J Mol Sci*. 2024;**25(16)**:8776. doi: 10.3390/ijms25168776.
5. El-Kamel MF, Sharaf MM, El-Anwar MH. Keloid and hypertrophic scars: comparative histopathological and immunohistochemical study. *Egypt J Dermatol Venerol*. 2014;**34(2)**:93-101. doi: 10.4103/1110-6530.150242.
6. Lee HJ, Jang YJ. Recent understandings of biology, prophylaxis and treatment strategies for hypertrophic scars and keloids. *Int J Mol Sci*. 2018;**19(3)**:711. doi: 10.3390/ijms19030711.
7. Morelli Coppola M, Salzillo R, Segreto F, Persichetti P. Triamcinolone acetonide intralesional injection for the treatment of keloid scars: patient selection and perspectives. *Clin Cosmet Investig Dermatol*. 2018;**11**:387-96. doi: 10.2147/CCID.S133672.
8. Gauglitz GG, Korting HC, Pavicic T, Ruzicka T, Jeschke MG. Hypertrophic scarring and keloids: pathomechanisms and current and emerging treatment strategies. *Mol Med*. 2011;**17(1–2)**:113-25. doi: 10.2119/molmed.2009.00153.
9. Jones CD, Guiot L, Samy M, Gorman M, Tehrani H. The use of chemotherapeutic agents for the treatment of keloid scars. *Dermatol Reports*. 2015;**7(2)**:5880. doi: 10.4081/dr.2015.5880.
10. Memariani H, Memariani M, Moravvej H, Shahidi-Dadras M. Emerging and novel therapies for keloids. *Sultan Qaboos Univ Med J*. 2021;**21(1)**:e22–33. doi: 10.18295/squmj.2021.21.01.004.
11. Abdel-Meguid AM, El-Kamel MF, Sharaf MM. Debulking of keloid combined with intralesional injection of methotrexate and triamcinolone versus intralesional injection of methotrexate and triamcinolone. *J Clin Dermatol Ther*. 2021;**7(1)**:033. doi: 10.24966/CDT-8771/100003.
12. Nagaria N. A comparative study of effectiveness of cryotherapy with intralesional triamcinolone vs fractional CO2 laser with topical betamethasone for the treatment of keloids [PhD thesis]. Kolar: Sri Devaraj Urs Academy of Higher Education and Research; 2022.
13. Walsh LA, Wu E, Pontes D, Kwan KR, Poondru S, Miller CH, *et al*. Keloid treatments: an evidence-based systematic review of recent advances. *Syst Rev*. 2023;**12(1)**:42. doi: 10.1186/s13643-023-02192-7.
14. Soliman M, Etman Y, AbdElhameed A, Elsharaby R, Tawfik A. Comparative study between Nd-YAG

laser, fractional CO2 laser, and combined Nd-YAG with fractional CO2 laser in the management of keloid: clinical and molecular study. *J Cosmet Dermatol*. 2021;**20(4)**:1124–32. doi: 10.1111/jocd.13920.

15. Elsaid D, Al-Tawel AA, Sabry H, Hasby E. Comparison between Er: Yag and Co2 ablative fractional lasers in the treatment of keloid and hypertrophic scars: histopathological, immuno-histochemical and ultrastructural study. *Benha J Appl Sci*. 2023;**8(2)**:45-56. doi: 10.21608/bjas.2023.188236.1038.
16. Bronte J, Zhou C, Vempati A, Tam C, Khong J, Hazany S, *et al*. A comprehensive review of non-surgical treatments for hypertrophic and keloid scars in skin of color. *Clin Cosmet Investig Dermatol*. 2024;**17**:1459–69. doi: 10.2147/CCID.S470997.
17. Alegre-Sánchez A, Jiménez-Gómez N, Boixeda P. Laser-assisted drug delivery. *Actas Dermo-Sifiliogr*. 2018;**109(10)**:858–67. doi: 10.1016/j.ad.2018.06.008.
18. Wang J, Wu J, Xu M, Gao Q, Chen B, Wang F, *et al*. Combination therapy of refractory keloid with ultrapulse fractional carbon dioxide (CO2) laser and topical triamcinolone in Asians-long-term prevention of keloid recurrence. *Dermatol Ther*. 2020;**33(6)**:e14359. doi: 10.1111/dth.14359.
19. Tawaranurak N, Pliensiri P, Tawaranurak K. Combination of fractional carbon dioxide laser and topical triamcinolone vs intralesional triamcinolone for keloid treatment: A randomised clinical trial. *Int Wound J*. 2022;19(7):1729-35. doi: 10.1111/iwj.13775.
20. Abd El-Dayem DH, Nada HA, Hanafy NS, Elsaie ML. Laser-assisted topical steroid application versus steroid injection for treating keloids: A split-side study. *J Cosmet Dermatol*. 2021;**20(1)**:138-42. doi: 10.1111/jocd.13521.
21. Park JH, Chun JY, Lee JH. Laser-assisted topical corticosteroid delivery for the treatment of keloids. Lasers Med Sci. 2017;**32(3)**:601–8. doi: 10.1007/s10103-017-2154-5.
22. Behrangi E, Jalilifar M, Lajevardi V, Razavi S, Azizian Z. Comparative effect of ablative fractional CO2 laser plus triamcinolone acetonide cream versus intralesional injection of triamcinolone acetonide in keloid and hypertrophic scars: A randomized clinical trial. *J Skin Stem Cell*. 2018;**5(1-2)**:e69394. doi: 10.5812/jssc.69394.
23. Alexander S, Girisha BS, Sripathi H, Noronha TM, Alva AC. Efficacy of fractional CO2 laser with intralesional steroid compared with intralesional steroid alone in the treatment of keloids and hypertrophic scars. *J Cosmet Dermatol*. 2019;**18(6)**:1648–56. doi: 10.1111/jocd.12887.
24. Shafqat A, Tahir K, Bashir F, Tahir M, Naveed A, Shaheen G. Comparison of efficacy of fractional CO2 laser with intralesional steroid versus intralesional steroid alone in the treatment of keloids. *J Pak Assoc Dermatol*. 2025;**35(3)**:221–6.